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in posture. An affirmative answer indicates that pericarditis is present; moreover, the electrocardiogram fails to show new changes which might result from extension of the myocardial lesion.

When chest pain is of pleuropericardial type or when pleural effusion is present, pulmonary infarction is an important differential diagnostic consideration. Evidence of fibrinous pericarditis or pericardial effusion, which is not usually a feature of pulmonary infarction,[10] supports the diagnosis of post-myocardial-infarction syndrome.

Recognition of the post-myocardial-infarction syndrome is important not only with regard to saving the patient and his relatives a good deal of mental anguish but also with regard to therapy. Anticoagulant therapy, which is used when extension of myocardial infarction or pulmonary infarction is diagnosed, is dangerous and contraindicated in the presence of generalized pericarditis.

Prognosis.—The outcome has been favorable in this series in all patients but the one who had been erroneously treated with anticoagulants although generalized pericarditis was diagnosed. The patient (Case 4) died of cardiac tamponade.

The complication is annoying because of pain and a tendency to relapses, but it is, on the whole, benign. When pain and cough are not a source of irritation, some patients feel strikingly well and request urgently to be allowed out of bed. To be sure, pericarditis may on occasions lead to cardiac tamponade even in the absence of anticoagulant effect, but this was not observed in the patients of this series.

Etiology.—As in the postcommissurotomy syndrome and in idiopathic pericarditis, the etiology of the syndrome complicating myocardial infarction is unknown. The severity of the infarction does not seem to be a factor, for the complication was observed both with massive infarctions and, less frequently, with mild myocardial lesions. In three instances of this series the acute infarction caused few symptoms and remained unrecognized. Yet in two of the patients a complication of marked degree developed. One sometimes wonders whether idiopathic pericarditis, especially when it affects older people, does not actually represent in some instances a post-myocardial-infarction syndrome, resulting from a small ischemic lesion of the myocardium which caused few or no symptoms and signs. The site of the myocardial infarction apparently does not exert a significant influence, for the syndrome was associated with equal numbers of anterior- and posterior-wall myocardial infarction.

It could be argued that anticoagulant therapy, by causing leakage of blood into the serous cavities, might produce symptoms and signs of pericarditis and pleuritis. Eleven patients of the group which was studied did not receive anticoagulants prior to the onset of the complication. Four of these were later treated with anticoagulants when the symptoms and signs of the syndrome were erroneously interpreted as due to extension of the myocardial infarction or to pulmonary infarction.

Attempts at culturing bacterial agents from blood and from pericardial- and pleural-aspiration fluid were unsuccessful; nor were we able to prove the presence of infection by serologic tests. Antibiotic drugs were ineffective. In this, as in many other respects, the complication of myocardial infarction resembles idiopathic pericarditis and the postcommissurotomy syndrome. A chance coincidence of idiopathic pericarditis and acute myocardial infarction is unlikely, since the incidence of the syndrome complicating myocardial infarction was about the same as that of idiopathic pericarditis alone, as observed in the Maimonides Hospital during the past three years.

Sensitization has been suggested as an etiological factor in idiopathic pericarditis and in the postcommissurotomy syndrome.[15,16] It has been shown in the experimental animal and in man that allergy might produce pericarditis, pleurisy, and